

- Handbook in Local Anaesthesia*, 2nd edn. London: Lloyd-Luke, 1979; 77–8
- 4 Goldberg MJ. Complication of cervical plexus block or fugue state? *Anesth Analg* 1995; **81**: 1108–9
 - 5 Stoneham MD, Wakefield TW. Acute respiratory distress after deep cervical plexus block. *J Cardiothorac Vasc Anesth* 1998; **12**: 197–8
 - 6 Szocik JF, Kellogg W, Wakefield TW. Temporary facial nerve palsy during carotid endarterectomy under local anesthesia. *Anesth Analg* 1995; **81**: 1106–7
 - 7 Lawrence PF, Alves JC, Jicha D, Bhirangi K, Dobrin PB. Incidence, timing and causes of cerebral ischemia during carotid endarterectomy with regional anesthesia. *J Vasc Surg* 1998; **27**: 329–34
 - 8 Castresana MR, Masters RD, Castresana EJ, Stefansson S, Shaker IJ, Newman WH. Incidence and clinical significance of hemidiaphragmatic paresis in patients undergoing carotid endarterectomy during cervical plexus block anesthesia. *J Neurosurg Anesthesiol* 1994; **6**: 21–3
 - 9 Davies MJ, Silbert BS, Scott DA, Cook RJ, Mooney PH. Superficial and deep cervical plexus block for carotid artery surgery. A prospective study of 1000 blocks. *Reg Anesth* 1997; **22**: 442–6
 - 10 Castresana MR, Brooks AG, Masters RD, Castresana EJ, Myers GJ, Newman WH. Incidence of dysphagia in patients undergoing carotid endarterectomy using deep and superficial cervical plexus block anesthesia. *Anesth Analg* 1994; **78**: S52
 - 11 Whitehurst L, Harrelson JM. Brain-stem anaesthesia. An unusual complication of stellate ganglion block. *J Bone Joint Surg (Am)* 1977; **59-A**: 541–2
 - 12 Ross S, Scarborough CD. Total spinal anaesthesia following brachial-plexus block. *Anesthesiology* 1973; **39**: 458
 - 13 Edde RR, Deutsch S. Cardiac arrest after interscalene brachial-plexus block. *Anesth Analg* 1977; **56**: 446–7
 - 14 Baraka A, Hanna M, Hammoud R. Unconsciousness and apnea complicating parascapular brachial plexus block: possible subarachnoid block. *Anesthesiology* 1992; **77**: 1046–7
 - 15 Tatum PL, Defalque RJ. Subarachnoid injection during retrobulbar block: a case report. *Am Assoc Nurse Anesth J* 1994; **62**: 49–52
 - 16 Mercereau DA. Brain stem anesthesia complicating retrobulbar block. *Can J Ophthalmol* 1989; **24**: 159–61
 - 17 Morgan GE. Post-retrobulbar apnea syndrome. *Reg Anesth* 1989; **14**: 203–5
 - 18 Kozody R, Ready LB, Barsa JE, Murphy TM. Dose requirement of local anaesthetic to produce grand mal seizure during stellate ganglion block. *Can Anaesth Soc J* 1982; **29**: 489–91
 - 19 Knudsen K, Beckman Suurkula M, Blomberg S, Sjoval J, Edvardsson N. Central nervous and cardiovascular effects of i.v. infusions of ropivacaine, bupivacaine and placebo in volunteers. *Br J Anaesth* 1997; **78**: 507–14
 - 20 Leoni A, Casati A, Fanelli G, Aldegheri G, Casaletti E, Torri G. A double-blind comparison between 0.75% ropivacaine and 2% mepivacaine for axillary brachial plexus anaesthesia. *Anaesth* 1998; **53**(Suppl 2): 92
 - 21 Winnie AP. Considerations concerning complications, side effects and untoward sequelae. In: Hakansson L, ed. *Plexus Anaesthesia: Perivascular Techniques of Brachial Plexus Block*. Philadelphia: WB Saunders, 1983; 221–65
 - 22 Norris D, Klahsen A, Milne B. Delayed bilateral spinal anaesthesia following interscalene brachial plexus block. *Can J Anaesth* 1996; **43**: 303–5
 - 23 Checketts MR, Wildsmith JAW. Accidental i.v. injection of local anaesthetics: an avoidable event? *Br J Anaesth* 1998; **80**: 710–1
 - 24 Winnie AP, Ramamurthy S, Durrani Z, Radonjic R. Interscalene cervical plexus block: a single-injection technic. *Anesth Analg* 1975; **54**: 370–5
 - 25 Scammell SJ. Inadvertent epidural anaesthesia as a complication of interscalene brachial plexus block. *Anaesth Intens Care* 1979; **7**: 56–7

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Transthoracic echocardiography for perioperative haemodynamic monitoring

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Transoesophageal echocardiography (TOE) is valuable for perioperative monitoring in patients at risk from haemodynamic disturbance. However, its use is not practicable in patients undergoing surgical procedures under regional anaesthesia. We describe two cases showing that transthoracic echocardiography (TTE) has the same advantages as TOE and thus may be valuable for monitoring awake patients. TTE should be considered when extended perioperative haemodynamic monitoring is needed but TOE is not possible.

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Transoesophageal echocardiography (TOE) is valuable for perioperative monitoring in patients at risk for haemodynamic disturbance.¹ When regional anaesthesia is chosen, as is often the case in patients undergoing Caesarean delivery, TOE is impracticable. We report two cases showing that transthoracic echocardiography (TTE) can image the heart during surgery and may be an alternative method of monitoring in such patients.

Case 1

A 34-yr-old, gravida 1, nulliparous woman was admitted at 15 weeks gestation with symptoms of congestive heart failure. She had no history of cardiovascular disease and early pregnancy was uneventful. TTE assessment showed dilated cardiomyopathy of unknown origin with impaired left ventricular (LV) systolic function (fractional shortening 21%) and bilateral pleural effusions. Despite treatment with digoxin, diuretics, nitrates and dihydralazine, orthopnoea and shortness of breath increased and fractional shortening decreased to 12%. Therefore, an urgent Caesarean delivery was planned at 32 weeks' gestation. No signs of foetal distress were present.

After arterial and central venous pressure monitoring had been started, a combined spinal-epidural anaesthesia (CSEA) technique was performed. Intrathecal injection of 0.5% hyperbaric bupivacaine 7.5 mg, fentanyl 10 µg and morphine 0.25 mg, supplemented by 0.5% bupivacaine 55 mg injected epidurally resulted in a T6 sensory level. At onset of anaesthesia, systolic arterial blood pressure transiently decreased from 140 to 85 mm Hg but then became steady around 95 mm Hg.

Baseline TTE (2.0/2.5 MHz probe, Sonos 2500, Hewlett Packard, Andover, MA, USA), which included parasternal long- and short-axis and apical four- and two-chamber views, was done before starting anaesthesia. The examination confirmed the preoperative findings of poor LV function and showed a fresh thrombus in the apex of the left ventricle, which had not been seen before (Fig. 1). As soon as CSEA had been started, LV dimensions and LV function (fractional shortening) were monitored in the parasternal long-axis view to control the effect of CSEA and to guide the quantity and speed of fluid replacement and administration of vasoactive drugs. The continuous qualitative assessment was supplemented by repeated quantitative measurements of LV end-diastolic and end-systolic dimensions and LV shortening fraction (Fig. 2). The strategy was to give intravenous fluids when arterial blood pressure and LV diastolic dimension decreased simultaneously, and to give vasoactive drugs when arterial pressure decreased without changes in LV diastolic dimension or with an

increase in LV systolic dimension (i.e. worsening of systolic function).

After starting CSEA, LV diastolic dimension decreased without changes in systolic shortening (Fig. 2), and the patient's shortness of breath improved. These observations were interpreted as a reduction in pre-existing hypervolaemia, and only minimal fluid was given. An estimated blood loss of 600 ml was replaced with 600 ml lactated

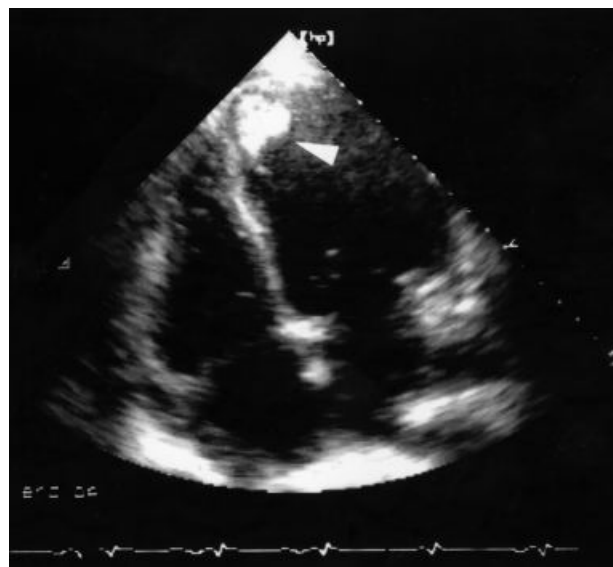


Fig 1 TTE of the patient of Case 1. Four-chamber view with apical thrombus in the left ventricle (arrowhead).

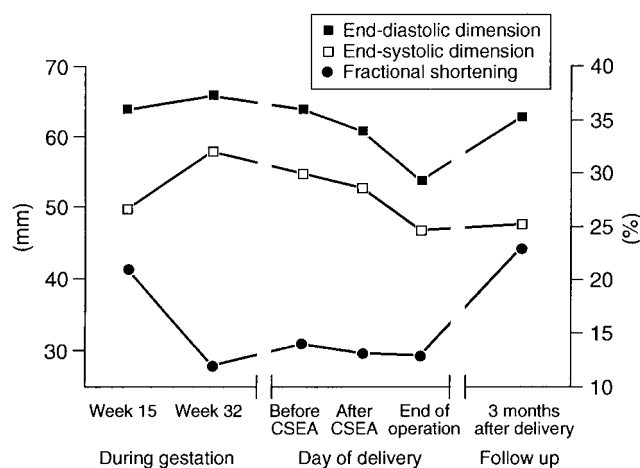


Fig 2 Course of end-diastolic and end-systolic dimensions and fractional shortening in the patient of Case 1.

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Ringer's solution, and a transient decrease in arterial blood pressure was treated with phenylephrine 100 µg.

Neonatal outcome was excellent. On the first postoperative day, the epidural catheter was removed after administration of morphine 3 mg. Anticoagulant therapy with heparin followed by coumarin was subsequently started.

The cardiological follow-up visit 3 months after delivery showed unchanged LV dimensions but improved LV function (fractional shortening 23%) (Fig. 2). The LV thrombus had disappeared and anticoagulant therapy was stopped.

Case 2

A 32-yr-old, gravida 3, para 2 woman was admitted at 39 weeks' gestation because of known cardiac disease. Six years before, bacterial endocarditis had caused severe mitral insufficiency needing surgical closure of a perforation in the posterior leaflet of the mitral valve.

The course of the actual pregnancy was uneventful and without any sign of congestive heart failure. Because of a systolic murmur, TTE was performed and showed severe mitral insufficiency, dilatation of the left atrium and left ventricle but preserved LV systolic function (fractional shortening 35%). An elective Caesarean delivery was planned. No signs of foetal distress were present.

After arterial and central venous pressure monitoring had been started, CSEA was given. Intrathecal injection of 0.5% hyperbaric bupivacaine 7.5 mg resulted in a T4 sensory level. At onset of anaesthesia, systolic blood arterial pressure transiently decreased from 125 to 90 mm Hg but then became steady around 120 mm Hg.

A complete TTE examination (2–4 MHz probe S4, Sonos 5500) was done when the patient came to the operating theatre. As soon as CSEA had been started, LV dimensions and function and the grade of mitral regurgitation were monitored continuously in the four-chamber view. The grade of mitral regurgitation was assessed semiquantitatively by comparing the area of colour Doppler jet with the left atrial area and the propagation of the jet into the left atrium. The management plan concerning fluids and the vasoactive drugs was the same as described in Case 1. The TTE guided haemodynamic management lactated Ringer's solution 2300 ml and hetastarch 500 ml, giving ephedrine 15 mg) caused no relevant changes in LV size or mitral regurgitation. The estimated blood loss was 600 ml.

Neonatal outcome was excellent. Postoperative analgesia was done by epidural administration of 0.1875% bupivacaine at the rate of 6 ml h⁻¹. On postoperative day one, the patient was in excellent clinical condition and TTE showed a smaller LV. After decreasing bupivacaine administration on day 2, the patient became dyspnoeic and oxygen saturation decreased to 88%. Clinical and radiological examination revealed acute pulmonary oedema.

After starting therapy with angiotensin-converting enzyme inhibitors, nitrates and diuretics, the patient's condition improved rapidly and she was discharged on the 10th postoperative day.

Discussion

According to the Practice Guidelines for Perioperative Transoesophageal Echocardiography, an increased risk of haemodynamic disturbance during the perioperative period is a category-II indication for perioperative TOE, indicating that 'TOE may be useful in improving clinical outcomes'.¹ Our two patients fulfilled these criteria, but the use of TOE was not feasible because they were awake. Regional anaesthesia was chosen because it is advantageous in patients undergoing Caesarean delivery who are at increased risk of aspiration of gastric contents.²

We show that TTE can be used instead of TOE for extended non-invasive haemodynamic monitoring in awake surgical patients, although the quality of the image may be more variable with TTE. TTE facilitated appropriate guidance of volume replacement and use of vasoactive drugs.

In Case 1, the echocardiographic finding of a decrease in LV end-diastolic dimension with unchanged systolic function after initiation of CSEA (Fig. 1), with a simultaneous decreased shortness of breath, led to the decision to replace blood loss only partially. In addition, the detection of a previously unknown LV thrombus markedly altered postoperative management.

In Case 2, echocardiographic findings contributed crucially to the decision to administer fluids in order to compensate for vasodilation caused by sympathetic blockade³ and for blood loss. Fluid replacement would have been stopped had LV dimensions increased, LV function worsened or mitral regurgitation increased.

As an alternative to TTE monitoring, we considered the use of a pulmonary artery catheter, which has been suggested for patients with significant cardiovascular disease who are at risk of haemodynamic disturbance.⁴ We decided against this because (1) it is more invasive than TTE, (2) echocardiography can provide superior information in haemodynamically unstable patients,^{5–7} and (3) direct visualization of the heart can provide clinically important new information (as with the thrombus in Case 1). Moreover, in critically ill patients the use of a pulmonary artery catheter has been associated with increased mortality in a recent study.⁸ In addition, using the same diagnostic technique before, during and after surgery can improve assessment of the course of the cardiac disease (Fig. 2). The importance of postoperative cardiovascular monitoring in cardiac risk patients is emphasized by the onset of acute pulmonary oedema on the second day, probably caused by reducing the epidural bupivacaine administration, decreasing the sympathetic blockade and increasing LV pre- and afterload.

TTE can be useful for non-invasive perioperative haemodynamic monitoring in patients with severe cardiac disease, and should be considered if extended perioperative haemodynamic monitoring is indicated but the use of TOE is impossible.

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References

- 1 Task Force on Perioperative Transesophageal Echocardiography. Practice guidelines for perioperative transesophageal echocardiography. *Anesthesiology* 1996; **84**: 986–1006
- 2 Santos AC, Pederson H, Finster M. Obstetric anesthesia. In: Barasch PG, Cullen BF, Stoelting RK, eds. *Clinical Anesthesia*. 3rd edn. Philadelphia: Lippincott-Raven, 1996: 1068–70
- 3 Lewis M, Thomas P, Wilkes RG. Hypotension during epidural analgesia for Caesarean section. Arterial and central venous pressure changes after acute intravenous loading with two litres of Hartman's solution. *Anaesthesia* 1983; **38**: 250–3
- 4 Practice guidelines for pulmonary artery catheterization. A report by the American Society of Anesthesiologists Task Force on Pulmonary Artery Catheterization. *Anesthesiology* 1993; **78**: 380–94
- 5 Sohn DW, Shin GJ, Oh JK, et al. Role of transesophageal echocardiography in hemodynamically unstable patients. *Mayo Clin Proc* 1995; **70**: 925–31
- 6 Reichert CL, Visser CA, Koolen JJ, et al. Transesophageal echocardiography in hypotensive patients after cardiac operations. Comparison with hemodynamic parameters. *J Thorac Cardiovasc Surg* 1992; **104**: 321–6
- 7 Fontes ML, Bellows W, Ngo L, Mangano DT. Assessment of ventricular function in critically ill patients: limitations of pulmonary artery catheterization. Institutions of the MCSPI Research Group. *J Cardiothorac Vasc Anesth* 1999; **13**: 521–7
- 8 Connors AF Jr, Speroff T, Dawson NV, et al. The effectiveness of right heart catheterization in the initial care of critically ill patients. SUPPORT Investigators. *J Am Med Assoc* 1996; **276**: 889–97

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Successful resuscitation from recurrent ventricular fibrillation secondary to butane inhalation

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Resuscitation from cardiac arrest caused by volatile substance abuse is rarely successful. Large doses of catecholamines given during resuscitation, in the presence of butane, may cause recurrent ventricular fibrillation. We report a case of prolonged resuscitation in a young man who had inhaled butane. Cardiac output was restored 10 min after the administration of intravenous amiodarone. We suggest that antiarrhythmic agents should be used early during resuscitation to prevent recurrent arrhythmias.

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Keywords: aerosols; complications, cardiac arrest, resuscitation; sympathetic nervous system, pharmacology, epinephrine

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Volatile substance abuse (VSA) may be defined as the deliberate inhalation of a volatile substance to achieve a change in mental state.¹ VSA is an important cause of death in those under 20 yr of age. In 1997, VSA accounted for 1 in 50 of all deaths (or 1.7 deaths per 100 000 population) in people aged 15–19 yr in the UK. Most of the deaths are caused by inhaled gas fuels. In 1997, 56% of all VSA deaths in the UK were associated with butane.² Butane is found mainly in cigarette lighter refills and is used as an aerosol propellant.

Case report

A 17-yr-old male with a 3-yr history of butane abuse was found collapsed in the street. By his side was a canister of butane lighter fuel. When the paramedical emergency team arrived he was in ventricular fibrillation (VF). They gave two cycles of the advanced cardiac life support protocol, which included epinephrine 2 mg. On arrival in the emergency department he had a carotid arterial pulse that was just palpable. He then developed VF again. Advanced life support continued for a further 40 min. During resuscitation